PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF

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Bacteriological and Experimental Studies on Gastric Ulcer.—Many studies and experiments have been undertaken to determine the pathological process in gastric ulcer. CELLER and THALHIMER (*Jour. Exper. Med.*, 1916, xxiii, 791), stimulated by the positive results reported by Rosenow, have repeated the work using his technic in isolating and injecting streptococci into rabbits and cats. The bacteria used in the experiments were isolated from the tissues of eight gastric ulcers and one ulcer at the ostium of a gastrojejunostomy. Histological examination of the human tissues showed the presence of organisms only in or upon the lining of the degenerating tissue of the ulcer. Various microörganisms were observed in this situation. In culture streptococci with other bacteria were isolated in 8, while a staphylococcus and Gram-positive bacillus were obtained in 1. Suspensions of the microorganisms obtained were inoculated into the ear vein or a branch of the gastric artery. In 2 cats a branch of the gastric artery was injected with streptococci. Both animals developed defects of the gastric mucosa which soon began to heal and were completely repaired in thirty-three days. Of the 30 rabbits injected intravenously with streptococci, gastric lesions developed in 4, while 14 showed cardiac lesions. In 8 rabbits a branch of the gastric artery was injected with streptococci and lesions developed in 6. The authors are not convinced that the lesions which they observed in their animals were truly gastric ulcers as there lacked the characteristics of those observed in man. The promptness of healing also strengthened the belief that the lesions in animals and man are not comparable. Embolic lesions in the stomachs of cats healed spontaneously and did not appear to be influenced by the presence of streptococci. They conclude that even though anhemolytic streptococci are present in practically all gastric ulcers it has not been proven that these microörganisms initiate the ulceration or prevent healing.

The Production of Amyloid Disease and Chronic Nephritis in Rabbits by Repeated Intravenous Injections of Living Colon Bacilli.—There is a considerable number of experiments on record in which amyloid has been induced in animals. For its production, bacteria of various kinds

as well as chemical substances were made use of. The positive results have been most frequently obtained in rabbits, but successful experiments have also been obtained in dogs, horses, fowl, and mice. BAILEY (*Jour. Exper. Med.*, 1916, xxii, 773) found amyloid degeneration in various organs of rabbits during an experimental study of the production of chronic nephritis by B. coli. Living cultures were injected every two to four days into rabbits. In a series of 15 animals positive results were obtained in 8. It was shown that time was an essential element, as no positive case was obtained under eighty-eight days, while every animal treated beyond this time showed amyloid. This product of tissue degeneration was not constant in its reaction to the various tests. It was found that whereas a positive result was obtained with methyl-violet or gentian violet, the iodin-sulphuric acid test was negative or indefinite. This lack in uniformity of the tests has been observed by others studying human material. The presence of amyloid was most commonly observed in the spleen, next the kidney, and finally the liver. As, in this series of experiments, suppuration was not induced in the tissues, it is evident that amyloid, though dependent upon the presence of bacterial growth, need not be associated with pus. The presence of amyloid in the kidney was associated with a subacute and chronic glomerulitis, parenchymatous degeneration, and some interstitial infiltration by round cells. Casts were present in tubules and a moderate fatty degeneration affected the convoluted tubules. The fatty deposit was not observed in association with the amyloid. No definite origin was determined for the amyloid deposit, but the author believed that it was of the nature of an infiltration rather than a production by either fibroblasts or endothelial cells.

Hematogenous Aortitis with Multiple Aneurysms.—Much the greater evidence upon the infection of arteries has been offered by French authors. This is more particularly true of infective arteritis associated with rheumatism. As early as 1840 Bouilland drew attention to the lesions in the heart and arteries as constituting an important phase in this disease. Brault, Stokes, Charcot, Vulpian, Corvisart, and others have described cases in which a more or less intense inflammation of the arterial walls was recognized and in some of which aneurysms had developed. Since 1900 at least 15 different French authors have reported cases and commented upon the presence of aneurysm associated with acute rheumatic fever. The majority of these cases were in young individuals ranging in age from three to twenty years. SOPRANA and PIAZZA (*Arch. de méd. exper.*, 1916, xxvii, 55) reported a case with multiple aneurysms of the aorta associated with rheumatism. The patient was a girl, aged twenty-one years, who had never had a previous attack of rheumatism. The fatal attack began suddenly and very acutely. She died within a month after the onset. During her illness she suffered from severe pain and swelling of the joints accompanied by fever. She also had much intrathoracic oppression with dyspnea. She died suddenly. At autopsy three aneurysms were found in the aorta, one of which had quite healthy looking walls and there was no evidence of damage upon the inner surface of the vessel. Microscopically the walls of each aneurysm showed much inflammatory infiltration as well as thickening of the adventitia. The media was much disturbed by the inflammatory processes with the destruction of

its essential elements, the muscle and elastic fibers. One aneurysm showed evidence of separation of the adventitia from the media thus forming a type of dissecting aneurysm. In this case the authors were unable to observe any evidence of organic syphilis and the Wassermann reaction was negative. They especially point out the fact that this patient had suffered her first attack of rheumatism during which the aneurysms had developed. They did not determine the nature of the microörganism which had led to tissue destruction. The aortic valves had been the seat of inflammatory reaction, but the infection associated with the aneurysm did not proceed directly along the aortic intima from the diseased valves. The authors believe that the infection of the aortic tissues had occurred by the carriage of the microörganisms to the adventitia. This is the more common mode of infection for these aortic lesions. The aneurysms develop only after the infection and its inflammation have brought about a certain amount of dissolution of the aortic wall.

Progressive Lenticular Degeneration.—New cases of Wilson's disease are periodically appearing in literature. As these cases are reported, the outstanding features of the disease are emphasized. Up to the present no one has analyzed the pathological findings to correlate them with the clinical picture. The constant presence of cirrhosis of the liver with degeneration of the nucleus lentiformis has not received adequate explanation. FARNELL and HARRINGTON (*Jour. Lab. and Clin. Med.*, 1916, 1, 561) have added another case to this interesting group. The patient was a girl of nineteen years who for four years had epileptoid seizures in which incoördination of the muscles of the extremities was one of the marked features. Her gait was somewhat spastic and at times her posture was fixed. Her mental development was below normal. At autopsy the striking finding was a marked cirrhosis of the liver of the portal type and the loss of the characteristic markings of the corpus striatum on the left side. The left caudate nucleus was smaller than normal and there was some dilatation of the left ventricle. The degeneration in the region of the nucleus lentiformis gave an appearance as if the various structures of this part were confluent. Small areas of necrosis were also present in this region. The microscopic examination of the affected brain tissues showed an increase of the glial tissue. The evidence of this degeneration was much more marked on the left than on the right side. There was no disturbance of the motor cortex or the cells of the anterior horns. The vessels were not sclerosed. The entire reaction appeared to be one of replacement gliosis which had not gone on to complete disintegration with cavity formation.

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